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CLINICAL RESEARCH

Intravascular Lithotripsy for Calcified Saphenous Vein Graft Stenoses in the Last Remaining Conduit: A Case Series and Review of the Literature

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ABSTRACT

Intravascular lithotripsy (IVL) is a safe and effective tool for calcium modification in coronary and peripheral vessels. IVL for treatment of calcified saphenous vein graft (SVG) lesions is an "off-label" indication and has not been well elucidated. Here we present successful use of IVL in two cases of SVG to left anterior descending (LAD) intervention. In both cases, the SVG was the major or last remaining conduit. Mechanical circulatory support (MCS) was used in one case but not in the other. We show that IVL is safe and effective for treatment of calcified SVG stenoses, even in situations where the SVG is the last remaining conduit, with or without the use of MCS.

INTRODUCTION

The greater saphenous vein was the primary graft used prior to 1986 for left anterior descending artery (LAD) territory surgical revascularization until the left internal mammary (LIMA) graft demonstrated a significant increase in graft patency and survival [1,2]. Repeat revascularization with percutaneous coronary intervention (PCI), either due to progression of native coronary artery disease or bypass graft failure, may be required in up to 20% of patients after coronary artery bypass grafting (CABG) [3]. Saphenous vein graft (SVG) patency rates continue to be poor with only 75% remaining patent at 1 year and 50%-60% remaining patent after a decade [3]. Mechanisms of late SVG failure include intimal hyperplasia leading to atherosclerosis which progresses at a much faster rate than native coronary artery atherosclerosis [3-5]. SVG PCI is fraught with issues such as high rate of no-reflow during PCI and higher rate of restenosis during follow-up [3]. Native vessel PCI is therefore preferred over SVG PCI when feasible [6]. However, due to accelerated native vessel atherosclerosis following CABG, up

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to 90% of patients with prior CABG have native vessel chronic total occlusions (CTOs), which may not be amenable to PCI [6,7].

Intravascular lithotripsy (IVL) is a novel therapy for treatment of vascular calcification. The safety and effectiveness of IVL for coronary and peripheral artery disease has been reported in several studies [8-14]. IVL uses acoustic shockwaves delivered through a semicompliant balloon inflated at 4 atmospheres (atm). IVL modifies both superficial and deep calcium by producing multiplane, circumferential and longitudinal calcium fractures, thereby allowing for adequate stent expansion [15]. The DISRUPT CAD I-IV studies included de-novo stenoses involving severely calcified coronary vessels [9,14,16,17]. None of these studies included calcified SVG lesions. There is limited data on the use of shockwave IVL in SVG lesions [18].

We present two cases of using shockwave IVL for calcified stenoses in SVG to LAD. It is notable that in both these cases the SVG to LAD was the major or last remaining conduit.

CASE DESCRIPTION

Case 1

A 73-year-old male with history of 4-vessel CABG in 1995 [SVG to LAD, SVG to ramus intermedius (RI), SVG to first diagonal and SVG to obtuse marginal (OM)-1], history of percutaneous coronary intervention to first diagonal branch with 2.75 x 8 mm Pixel stent in 2003, chronic heart failure with reduced ejection fraction [HFrEF, left ventricular ejection fraction (EF) 20-25%], history of dual chamber implantable cardioverter defibrillator (ICD) placement, non-insulin dependent diabetes mellitus, chronic obstructive pulmonary disease (COPD), paroxysmal atrial fibrillation on long term anticoagulation with apixaban and recurrent esophageal strictures was admitted for esophageal stricture dilatation. Post-procedure, he developed hypoxic respiratory failure requiring endotracheal intubation. Following this, the patient had a prolonged hospital course complicated by reintubation and aspiration pneumonia. During this period, the patient had non-ST elevation myocardial infarction (NSTEMI) with peak troponin-I value of 0.98 and dynamic ST depressions and diffuse T-wave inversions in inferolateral leads.

He underwent cardiac catheterization which showed multivessel CAD with heavily calcified, diffuse CTO of mid LAD after a large first diagonal branch, CTO of mid left circumflex with left to left and right to left collaterals filling the distal OM and left posterolateral branches and CTO of distal right coronary artery (Supplementary Video 1). SVG to OM-1 was patent. SVG to first diagonal and SVG to ramus intermedius were chronically occluded at the origin. SVG to LAD showed severe calcified 80% stenosis in the mid portion (Figure 1A, Supplementary video 1). Native LAD beyond the SVG provided left to right collaterals to right posterior descending artery (RPDA).

After stabilization of his respiratory issues and pneumonia, the patient was referred for complex and high-risk PCI of SVG to LAD. Decision was made to use mechanical circulatory support (MCS) with Impella CP® (Abiomed) device given large vascular territory supplied by SVG to LAD (which supplied collaterals to RPDA), intervention on an 18-year-old vein graft and poor left ventricular reserve with EF 20-25%. Bilateral femoral arterial access was obtained. Right femoral venous access was obtained, and right heart catheterization was performed which showed normal filling pressures, normal pulmonary pressures and preserved cardiac index. Bilateral iliofemoral angiogram showed adequate iliofemoral access for large bore MCS device. Preclosure of left femoral arterial

access was performed using two Perclose ProGlide™ closure devices (Abbott Vascular) at 10 o'clock and 2 o'clock positions. Via this access, an Impella CP® device was advanced into the left ventricle for hemodynamic support during the intervention.

The SVG to LAD was engaged using a 6 Fr multipurpose guide catheter and the lesion was wired using an 0.014" workhorse wire [Runthrough™ NS (Terumo)]. Distal embolic protection was attempted using a SpiderFX™ (Medtronic) 5.0 mm embolic protection device (EPD). However, given the calcified nature of the SVG stenosis, no equipment could be delivered over the filter wire without backing the filter out; therefore, EPD was removed and Runthrough™ wire was reinserted. A 6 Fr guideliner was inserted for additional support. The lesion was predilated using a 3.0 mm noncompliant balloon. Following this, intravascular ultrasound (IVUS) was performed using Opticross™ HD IVUS catheter (Boston Scientific). IVUS showed a large (5.5 mm) vein graft with severe 360-degree calcification and dense, eccentric calcification with severe stenosis at the site of the culprit lesion (Figure 1B). The minimal luminal area (MLA) was 3.4 mm^2 (Figure 1B). Following this, cutting balloon atherotomy was performed with a 4.0 x 6 mm Wolverine™ balloon (Boston Scientific). Despite high pressure inflation, there was a persistent waist ("dog-bone") noted in the balloon (Figure 1C). At this point, the decision was made to perform intravascular lithotripsy using the largest available (4.0 x 12 mm) coronary Shockwave™ IVL balloon (Shockwave Medical). The Shockwave balloon was inflated to 4 to 6 atm at the lesion site in mid portion of SVG to LAD and all 80 pulses were delivered. Following this, the 4.0 mm Shockwave balloon was inflated at 18 atm with no waist visible on fluoroscopy. IVUS revealed luminal gain and fractures in the calcium at the lesion site (Figure 1 D). Additional predilation of the SVG lesion was performed with a 4.5 x 15 mm NC balloon inflated at 18 atm. Following this, PCI of SVG to LAD was performed with overlapping 5.0 x 24 and 5.0 x 8 mm Synergy™ Megatron (Boston Scientific) drug eluting stents (DES). The stents were post dilated with a 5.0 mm noncompliant balloon at 18 atm. Final IVUS showed good stent expansion and excellent apposition with minimal stent area (MSA) 13.1 mm^2 (Figure 1E). The final angiogram showed excellent angiographic results with no evidence of vessel perforation or dissection and thrombolysis in myocardial infarction (TIMI)-3 flow (Figure 1F). At the end of the procedure, Impella CP® was weaned and removed and the large bore access was successfully closed using the previously placed suture-mediated closure devices with excellent hemostasis.

Post-procedure the patient had a prolonged hospitalization complicated by several non-cardiac issues and underwent percutaneous endoscopic gastrostomy tube placement due to dysphagia and esophageal stenosis as well as tracheostomy due to recurrent respiratory failure. Ultimately, he was discharged home without requiring oxygen and with close follow-up with home health visits. He was maintained on apixaban in combination with clopidogrel at the time of discharge. At three months follow-up, he reported that he was getting better and offered no cardiac complaints.

Case 2

A 73-year-old female with history of CABG in 1996 with SVG to LAD and SVG to left circumflex (LCX) OM branch, right internal carotid artery stenosis status post endarterectomy, severe peripheral arterial disease (PAD) status post left common femoral endarterectomy and femoral to above knee popliteal artery bypass as well as right common femoral endarterectomy and right profundoplasty and history of nephrectomy (kidney transplant donor in 1991) underwent a pharmacologic nuclear stress test prior to risk stratification for left carotid endarterectomy. The nuclear stress test revealed high-risk findings including transient ischemic dilatation, hence the patient was

referred for coronary angiography. Preoperative echocardiogram revealed normal left ventricular (LV) function with EF 55%.

Coronary angiogram revealed severe native vessel CAD with CTO of ostial left main (LM) and ostial right coronary artery (RCA). Native LM and RCA could not be engaged with multiple diagnostic catheters since they were occluded. SVG to OM1 branch of LCX was patent. However, OM2 branch of LCX was 100% occluded distally and filled via left-to-left collaterals from the LAD (that filled via SVG). There were left-to-left collaterals from OM1 to the first diagonal branch (Supplementary Video 2). SVG to LAD had a patent stent in the proximal segment following which there was de-novo 95% tubular, heavily calcified stenosis in the mid segment. Native LAD beyond the anastomosis had a patent stent and supplied left-to-left collaterals to the LCX OM2 branch and left-to-right collaterals to the RCA (Figure 2A, Supplementary Video 2).

Complex high-risk coronary intervention was planned on the SVG to LAD. The decision was made not to use MCS given normal LV function, no clinical evidence of decompensated heart failure and presence of severe PAD that would limit MCS options and increase risk of vascular complications. SVG to LAD was engaged using a 6 Fr internal mammary guide and wired using a 0.014" workhorse coronary guidewire [Runthrough^{TM} NS (Terumo)]. SVG-LAD had a balloon undilatable lesion despite high pressure inflation with a 3.5 mm non-compliant balloon (Figure 2B). The Opticross^{TM} HD (Boston Scientific) IVUS catheter did not cross the lesion despite use of a 6 Fr guideliner for additional support, hence IVUS was not performed. Subsequently, IVL was performed using 3.5 x 12 mm Shockwave^{TM} IVL balloon (Shockwave Medical) at 4 to 6 atm delivering all 80 pulses. Following this, high pressure balloon angioplasty was performed using a 3.5 mm noncompliant balloon at 18 atm with adequate expansion at the lesion site and disappearance of the waist on fluoroscopy (Figure 2C). Subsequently, PCI of SVG to LAD was performed using 4.0 x 20 mm Promus Elite drug eluting stent that was post dilated with 4.0 mm noncompliant balloon at 18 atm. Final angiogram showed excellent angiographic results with no vessel perforation or dissection and TIMI-3 flow (Figure 2D).

The patient was tolerated the procedure well without any issues and was discharged on the next postoperative day. The patient was placed on dual antiplatelet therapy (DAPT) with aspirin and clopidogrel. The patient was doing well on outpatient follow-up at 8 weeks with no anginal symptoms. She is scheduled for planned elective vascular surgery (left carotid endarterectomy) in a few weeks.

DISCUSSION

To our knowledge, this case series is the first description of IVL use for calcified stenoses in SVG to LAD wherein the vein graft was the major or last remaining conduit. We show that IVL use in SVG was safe and effective and allowed for adequate stent expansion. We used MCS in the first case (EF 20-25%) but not in the second case (normal EF, severe PAD). In both cases, the patients tolerated the procedure well without any complications (e.g., no-reflow). Case 2 illustrates that the use of IVL in calcified SVG stenosis is safe without the use of MCS despite the SVG being the last remaining conduit, especially if the baseline LV function is normal.

The use of MCS with Impella CP® during complex, high-risk PCI of heavily calcified unprotected left main [19,20] or last remaining coronary artery [21,22] has been elucidated in prior reports. The prior iteration of the IVL balloon had a total of 80 pulses and the new iteration of the balloon has a total of 120 pulses of IVL with a single cycle involving 10 IVL pulses. At least 10 seconds of balloon inflation is required to deliver a full cycle of 10 IVL pulses. Depending upon the amount of calcium modification required, all 80 to 120 pulses may need to

be used, requiring a total of 8 to 12 cycles of at least 10 seconds of inflation. The goal of circulatory support in these situations is to avoid severe hypotension during prolonged inflations of the Shockwave IVL balloon in unprotected LM or last remaining coronary vessel, especially in the setting of severe LV dysfunction [20]. Based on this principle, we elected to use MCS in our first case (Case 1) given that the LAD distal to the SVG anastomosis provided left to right collaterals to the RPDA and the patient had severe baseline LV dysfunction with EF 20-25%. The second case (Case 2) would have been ideal for MCS use as well; however, with normal baseline LV function and severe PAD, the risks of MCS (e.g., vascular complications) outweighed potential benefits.

Native vessel PCI post CABG, although preferred [6], may not be feasible in those with complex anatomy and extensive CTOs post CABG. In certain cases, SVG PCI may be the only viable option despite issues associated with intervention in old and degenerated vein grafts. Rarely, SVG atherosclerosis may be accompanied by severe calcification. In contrast to native coronary artery calcification, SVG calcification is not limited to plaque but occurs predominantly in the vessel wall [23]. This is a result of arterialization of the graft due to chronic hemodynamic stress [23]. The presence of significant SVG calcification may necessitate calcium modification techniques such as atherectomy and IVL. Both rotational and orbital atherectomy have been used “off-label” for calcified, undilatable SVG lesions [24-29]. Rotational atherectomy has been shown to be safe and effective for both de-novo SVG lesions [24,26,27,29] as well as in-stent restenosis (ISR) [28]. With the advent of IVL, it is prudent to think that IVL can be an effective alternative in calcified SVG lesions that would have otherwise required atherectomy. There are several prior reports of IVL use for calcified de-novo as well as in-stent SVG stenoses [18,26,30-34]. Meijer et al. [34] reported successful use of IVL in a series of 4 patients with peri-stent calcific lesions in SVGs both in the setting of calcific in-stent neoatherosclerosis (2 cases) and as bailout acutely in the setting of stent under-expansion due to extrinsic calcification (2 cases). Table 1 summarizes prior reports of IVL use in SVG and includes 14 SVG lesions (across 7 studies) with 6 being de-novo lesions and 8 being ISR. Rotational atherectomy was used as adjunct therapy prior to IVL use in one case of de-novo calcified SVG stenosis [26], whereas drug-eluting balloon was used post-IVL in another case involving recurrent ISR [18]. MCS was not used in any study. In all studies, IVL was effective, was not associated with any complications and most patients were doing well in the follow-up period (Table 1).

A theoretical concern with IVL use in SVG is risk of slow-flow/no-reflow due to multiple balloon inflations that would be required with IVL use. Prior studies have identified degenerated vein grafts, presence of thrombus or intraluminal mass, lesion length, stent length, reduced LV function, and age of CABG as risk factors that predict slow-flow/no-reflow after SVG PCI [35-40]. SVG degeneration is defined as luminal irregularities or ectasia involving 50% or more of the length of the graft [39,41], i.e., diffusely diseased vein grafts. There is limited data on SVG lesion characteristics on IVUS that predict slow flow/no-reflow after SVG PCI [35,38]. In a study looking at virtual history imaging in SVG lesions [35], the SVG plaque morphology was classified into 4 types: fibrous tissue, fibrofatty, necrotic core and dense calcium. This study showed that only the absolute necrotic core area was independently predictive of slow-flow/no-reflow and major adverse cardiovascular events after SVG PCI [35]. It can be therefore conjectured that SVG lesions with dense calcification may be at a lower risk of slow-flow/no-reflow, making IVL a relatively safe treatment option; however, there are no data to support this conjecture.

In our case series, the factors favouring safe use of IVL with relatively low risk of slow-flow/no-reflow were short lesion lengths and absence of degeneration in remaining portion of the SVG (Figures 1 and Figure 2). Our report is unique since it involves IVL use in calcified SVGs that were the last remaining conduits. We show that IVL use in this scenario is safe and effective.

CONCLUSIONS

We report two cases of IVL use in calcified de-novo SVG to LAD stenoses wherein the SVG was the major or last remaining conduit. We show that IVL is a safe and effective tool for calcium modification in calcified vein graft stenoses.

REFERENCES

1. Loop FD, Lytle BW, Cosgrove DM et al. (1986) Influence of the internal-mammary-artery graft on 10-year survival and other cardiac events. The New England Journal of Medicine 314(1): 1-6.
2. Goldman S, Zadina K, Moritz T et al. (2004) Long-term patency of saphenous vein and left internal mammary artery grafts after coronary artery bypass surgery: results from a Department of Veterans Affairs Cooperative Study. Journal of American College of Cardiology 44(11): 2149-56.
3. Xenogiannis I, Zenati M, Bhatt DL et al. (2021) Saphenous vein graft failure: From pathophysiology to prevention and treatment strategies. Circulation 144(9): 728-745.
4. Yazdani SK, Farb A, Nakano M et al. (2012) Pathology of drug-eluting versus bare-metal stents in saphenous vein bypass graft lesions. JACC Cardiovascular Intervention 5(6): 666-674.
5. Song YQ, Xu Y, Guo ZG (2020) Risk factors and possible mechanisms of saphenous vein graft failure after coronary artery bypass surgery. Chinese Medicine Journal (Engl) 133(13): 1606-1608.
6. Lawton JS, Tamis-Holland JE, Bangalore S et al. (2022) 2021 ACC/AHA/SCAI guideline for coronary artery revascularization: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation 145(3): e18-e114.
7. Jeroudi OM, Alomar ME, Michael TT et al. (2014) Prevalence and management of coronary chronic total occlusions in a tertiary Veterans Affairs hospital. Catheter Cardiovascular Intervention 84(4): 637-643.
8. Ali ZA, McEntegart M, Hill JM et al. (2020) Intravascular lithotripsy for treatment of stent underexpansion secondary to severe coronary calcification. European Heart Journal 41(3): 485-486.
9. Ali ZA, Nef H, Escaned J et al. (2019) Safety and effectiveness of coronary intravascular lithotripsy for treatment of severely calcified coronary stenoses: The disrupt CAD II study. Circular Cardiovascular Intervention 12(10): e008434.
10. Brodmann M, Holden A, Zeller T (2018) Safety and feasibility of intravascular lithotripsy for treatment of below-the-knee arterial stenoses. Journal of Endovascular Therapy 25(4): 499-503.
11. Brodmann M, Schwindt A, Argyriou A et al. (2019) Safety and feasibility of intravascular lithotripsy for treatment of common femoral artery stenoses. Journal of Endovascular Therapy 26(3): 283-287.
12. Brodmann M, Werner M, Brinton TJ et al. (2017) Safety and performance of lithoplasty for treatment of calcified peripheral artery lesions. Journal of American College of Cardiology 70(7): 908-910.
13. Brodmann M, Werner M, Holden A et al. (2019) Primary outcomes and mechanism of action of intravascular lithotripsy in calcified, femoropopliteal lesions: Results of disrupt PAD II. Catheter Cardiovascular Intervention 93(2): 335-342.
14. Hill JM, Kereiakes DJ, Shlofmitz RA et al. (2020) Intravascular lithotripsy for treatment of severely calcified coronary artery disease. Journal American College of Cardiology 76(22): 2635-2646.

15. Kereiakes DJ, Virmani R, Hokama JY et al. (2021) Principles of intravascular lithotripsy for calcific plaque modification. JACC Cardiovascular Intervention 14(12): 1275-1292.
16. Brinton TJ, Ali ZA, Hill JM et al. (2019) feasibility of shockwave coronary intravascular lithotripsy for the treatment of calcified coronary stenoses. Circulation 139(6): 834-836.
17. Saito S, Yamazaki S, Takahashi A et al. (2021) Intravascular lithotripsy for vessel preparation in severely calcified coronary arteries prior to stent placement - primary outcomes from the japanese disrupt CAD IV Study. Circulation Journal 85(6): 826-833.
18. Oksnes A, McEntegart M (2021) Intravascular lithotripsy in saphenous vein grafts and graft stent failure: A case series. Catheter and Cardiovascular Intervention 97(7): E945-E950.
19. Agrawal Y, Zoltowska D, Nazroo JR et al. (2021) Impella-assisted intracoronary lithotripsy of severely calcified left main coronary artery bifurcation for NSTEMI with cardiogenic shock. Cureus 13(4): e14772.
20. Ristalli F, Maiani S, Mattesini A et al. (2020) Intravascular lithotripsy and impella support to assist complex lm angioplasty. Cardiovascular and Revascular Medicine 21(11S): 143-146.
21. Rola P, Wlodarczak A, Jastrzebski A et al. (2021) Impella protected percutaneous coronary intervention on the last remaining highly calcified coronary artery facilitated by shockwave intravascular lithotripsy and levosimendan infusion. Kardiology Pol 79(10): 1145-1146.
22. Chan KCA, Luk NHV, Lee KYM et al. (2019) A Case of rota-shock-pella. JACC Case Report 1(5): 765-770.
23. Castagna MT, Mintz GS, Ohlmann P et al. (2005) Incidence, location, magnitude, and clinical correlates of saphenous vein graft calcification: an intravascular ultrasound and angiographic study. Circulation 111(9): 1148-1152.
24. Don CW, Palacios I, Rosenfield K (2009) Use of rotational atherectomy in the body of a saphenous vein coronary graft. Journal of Invasive Cardiology 21(9): E168-170.
25. Gedela M, Li S, Bhatnagar U et al. (2020) Orbital atherectomy and heavily calcified saphenous vein graft intervention. Tex Heart Institute J 47(1): 41-43.
26. Bawamia B, Williams P (2021) Combined rotational atherectomy and intravascular lithotripsy to treat a calcified vein graft stenosis. Cardiovascular and Revascular Medicine 28S: 201-202.
27. Coto H (1998) Intravascular ultrasound-guided rotational atherectomy and stent implant in a previously undilatable saphenous vein graft lesion. Journal of Invasive Cardiology 10(8): 451-453.
28. Ponangi UP, Baveja S (2019) Successful angioplasty of saphenous venous graft in-stent restenosis using rotational atherectomy. BMJ Case Report 12(10): e231331.
29. Pellicano M, Flore V, Barbato E et al. (2018) From debulking to delivery: sequential use of rotational atherectomy and Guidezilla for complex saphenous vein grafts intervention. BMC Cardiovascular Disorder 18(1): 122.
30. van Gameren M, Vos J, IJ AJJ et al. (2020) Use of lithotripsy in a calcified saphenous vein graft. JACC Case Rep 2(12): 1891-1894.
31. Russo F, Chieffo A, Carlino M et al. (2021) Intravascular ultrasound-guided coronary lithotripsy treatment of in-stent restenosis in saphenous venous graft. Journal of Invasive Cardiology 33(2): E141-E142.
32. Wanczura PM, Stecko WB (2021) Intravascular lithotripsy in recurrent restenosis in underexpanded saphenous vein graft stent. Postepy Kardiol Interwencyjnej 17(4): 419-420.
33. Iwanczyk S, Skorupski W, Klotzka A et al. (2021) Intravascular ultrasound-guided lithotripsy in a calcified saphenous vein graft. Kardiol Pol 79(11): 1290-1291.

34. Meijer M, Oliveri F, van Oort MJH et al. (2025) Intravascular lithotripsy for the treatment of peri-stent calcific lesions in saphenous vein grafts: A case series report. Cardiovascular and Revascular Medicine 70: 85-91.
35. Liu Y, Li CP, Wang YY et al. (2020) Prediction of Major Adverse Cardiovascular Events and Slow/No-Reflow by Virtual Histology Imaging After Percutaneous Interventions on Saphenous Vein Grafts. Angiology 71(3): 263-273.
36. Kanal Y, Seyda Kanal HE, Yakut I et al. (2023) CRP Albumin Ratio May Predict No Reflow in Patients Undergoing Percutaneous Coronary Intervention for Saphenous Vein Graft Stenosis. Angiology 74(1): 55-61.
37. Gurbak I, Panc C, Sahin AA et al. (2020) CHA2DS2-VASc score as a predictor of no-reflow phenomenon after saphenous vein graft percutaneous coronary intervention in patients with non-ST-segment elevation acute coronary syndromes. Kardiol Pol 78(11): 1129-1136.
38. Hong YJ, Jeong MH, Ahn Y et al. (2012) Intravascular ultrasound findings that are predictive of no reflow after percutaneous coronary intervention for saphenous vein graft disease. American Journal of Cardiology 109(11): 1576-1581.
39. Sdringola S, Assali AR, Ghani M et al. (2001) Risk assessment of slow or no-reflow phenomenon in aortocoronary vein graft percutaneous intervention. Catheter Cardiovascular Intervention 54(3): 318-324.
40. Hashemi-Jazi M, Hosseini SM, Gholamrezaei A (2017) Factors associated with the no-reflow phenomenon following percutaneous intervention of saphenous vein coronary bypass grafts. ARYA Atherosclerosis 13(5): 221-229.
41. Coolong A, Baim DS, Kuntz RE et al. (2008) Saphenous vein graft stenting and major adverse cardiac events: a predictive model derived from a pooled analysis of 3958 patients. Circulation 117(6): 790-797.